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BY

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THE occurrence of cirrhosis of the liver in the lower animals has, so far as I am aware, been little studied. Some observers have gone so far as to deny its existence in animals other than man.¹ But it is well known, from experiments to which I shall later allude, that one form of cirrhosis may be produced by ligature of the common bile-duct. My present purpose is merely to place on record two cases. Their study is also of interest, as throwing light upon some points in the pathology and clinical history of cirrhosis.

CASE I. In 1878 I had, through the kindness of Dr H. R. Hutton, an opportunity of examining the liver of a cat which died with ascites. The cat belonged to a hospital porter who was popularly credited with alcoholic excess, but there was no evidence that the cat was also allowed to indulge. The liver was greatly enlarged, smooth on the surface, somewhat firm. On section it was very pale, the lobules apparently separated by pale infiltration, and their outlines obscured; although the individual lobules were readily distinguished by the position of their central veins, and their general arrangement was not distorted.

There was no general icterus, nor was the liver at all bile-stained. The ascitic fluid was also free from trace of bile-staining.

The naked-eye appearance of the organ suggested that of a leucocythæmic liver.

Microscopic examination showed this view to be erroneous. The central vein of each lobule was distinct, and in some cases dilated. The outline of the lobules was very irregular, the liver cells at the periphery separated either by small-celled infiltration, or by delicate strands of connective tissue. Wide bands of connective tissue, equalling in many places the width of the lobules, separated them from one another. But the most striking fact was the presence, in the connective tissue, of dense plexuses of young bile-ducts and bile-capillaries, which formed a considerable part of the tissue in some places.

¹ Thus Dr Clifford Allbutt in his admirable "Address in Medicine" (*Brit. Med. Jour.*, Aug. 11, 1888, p. 290), at the British Medical Association, says, "In any animal other than man cirrhosis of the liver is equally unknown." Dr Allbutt was, however, evidently misled in this, as in some other analogous assertions, by placing too implicit confidence in the statements of one observer.

These bile-capillaries could be traced in many places to the end of a row of liver cells, and their formation appeared in part to be effected by fission of the liver cells, in such a manner that the epithelium of the bile-duct was formed by the divided liver cell, whilst its channel probably corresponded with the bile canaliculus. (See Fig. 1.)



FIG. 1.

The views which I then expressed as to the nature of the anastomosing double rows of cells, and of their relation to the original bile-ducts and liver cells respectively, which would, I suppose, now be concurred in by all or nearly all pathologists, did not find favour with some of my pathological friends. They maintained that they were either atrophied liver cells, or abortive attempts at the formation of simulated bile-ducts, unconnected with the real bile-ducts. This opposition was fortunate, for it led me to determine, at the earliest opportunity, to test the fact by injecting the common bile-duct.

At the Brown Institution (with which I was then connected as Professor-Superintendent) there were frequent opportunities for obtaining diseased animals, which were often brought there by their owners to be killed. I therefore requested my colleague, Mr George Banham, F.R.C.V.S., then Veterinary Surgeon to the Institution, to look out for any cat presenting similar symptoms, especially ascites. This he was good enough to do, and very shortly afterwards with success.

CASE II. A large cat was brought to be killed, suffering from great swelling of the abdomen, which on examination proved to be ascitic. No history of the disease could be obtained, except that the condition

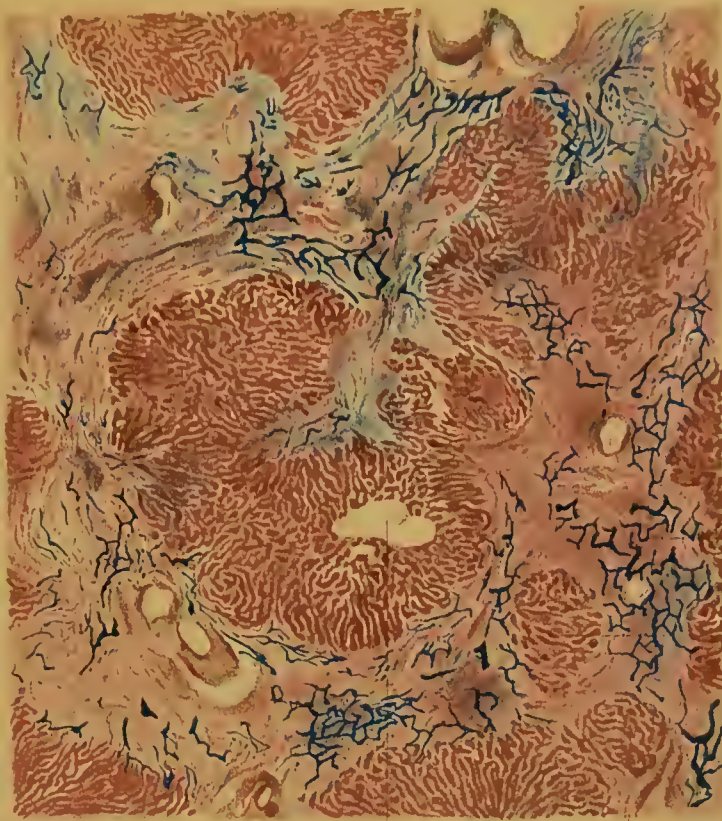


FIG. 2.

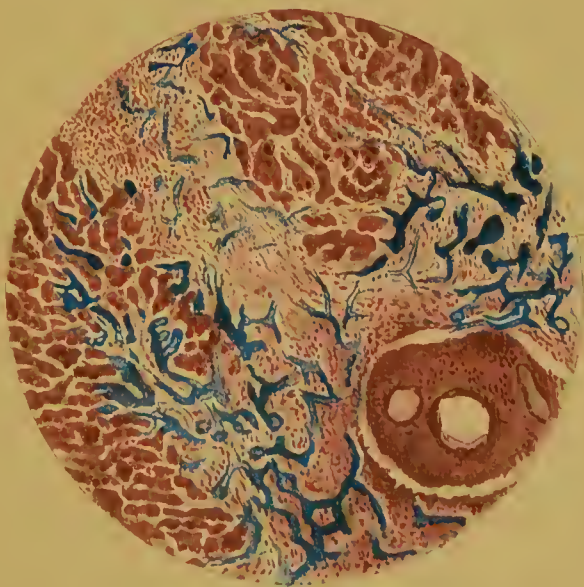


FIG. 3.



had come on gradually. The liver could be felt to be enlarged and firm. There was no trace of jaundice.

I at once made preparations for injecting the bile-ducts, and having killed the cat with chloroform, I exposed the common bile-duct, and at once injected it with Berlin blue. I had intended also to inject the portal vein whilst the liver was *in situ*, and therefore disturbed the organ as little as possible, so that no complete observation of its appearance was made until after injection and removal from the body. (The injection of the portal vein was not carried out.)

After removal the organ was found to be greatly enlarged, firm, and smooth. The ascitic fluid was in very large quantity, not bile-stained, it presented no special characters.

To avoid all risk of error, I injected in the same manner the liver of a cat which had to be killed for a tumour, and was thus able to compare the two, both with the naked eye and the microscope. An enormous difference in colour was at once perceptible, the cirrhotic liver being very deeply injected throughout, the healthy liver showing very little colour on the surface.

The other viscera were normal in appearance.

Microscopic examination showed the condition of the liver to be much the same as in the previous case, but there were one or two striking differences. The cirrhosis, as in Case I., was mono-lobular, that is, each lobule was separated by a wide area of connective tissue from the others. The connective tissue was, however, younger, *i.e.* more cellular, and was in larger amount. The plexuses of bile-capillaries were more abundant, and in many places occupied a large part of the interlobular connective tissue. (See *b*, Fig. 3.) Their epithelium was distinct throughout, as in the previous case. (It is not so distinctly seen in the drawings, as it is somewhat obscured by the injection, and the drawings are made with lower powers.)

The masses of liver cells remaining unaffected were somewhat more sharply defined than in Case I., that is, they more closely resembled the condition in common alcoholic cirrhosis, and were not so much frayed out at the edge by penetration of the connective tissue strands. But they were not in any marked degree crowded together or distorted.

The other very striking fact was the condition of the vessels. The central veins of the lobules were in some places markedly dilated, and there was very marked dilatation of the intralobular capillaries. In the connective tissue bands there was also in some parts much dilatation of the vessels, but there was little evidence of such newly formed vessels as are frequently seen in this position in cirrhosis. The larger bile-ducts appeared quite normal. The liver cells which remained showed little or no morbid change.

In some parts of the liver, where the injection had not penetrated, the newly-formed bile-ducts could be more fully studied. Each had a distinct but delicate membranous wall, and was lined by a single layer of cubical epithelium, which in most occupied a great part of the tube, leaving only a small central lumen. The apparent mode of formation was the same as in the previous case.

It is perhaps desirable to add that comparison with the healthy injected cat's liver left no doubt that the plexuses of bile-capillaries and ducts were of new formation. In the liver of the cat the arrange-

ment and proportion of the interlobular bile-ducts and capillaries differs but little from that in the human liver, and the lobules are not more separate, as they are in the pig. I regret that I have no drawing prepared from the healthy cat's liver by way of comparison; the contrast is a most striking one.

A few remarks upon the principal conclusions to be drawn from these two cases must suffice.

They place beyond doubt the occurrence, and, one might almost conclude, the frequency of cirrhosis of the liver in domestic cats. They throw no light upon the etiology of the disease. The form of cirrhosis is one, which though not common in the human subject, is closely analogous to conditions frequently observed in a partial degree in many instances of cirrhosis.

But they have also a bearing upon some of the disputed points in relation to human cirrhosis. It would not be fitting that I should here enter into a full discussion of a subject which is chiefly of interest in relation to human clinical medicine. Yet some of the facts are of a wider interest, and I will therefore give a very brief statement of those of most importance.

Common cirrhosis in man, in the form attributed to alcoholic excess, usually produces ascites, often to a great degree, is accompanied by very slight jaundice, often so slight as to be almost imperceptible, rarely pronounced. The liver, whether enlarged, or, as usual, smaller than normal, is irregular or granular on the surface. The connective tissue is formed in excess especially round certain of the larger portal venules, and therefore includes groups of lobules rather than individual lobules, hence this form has been called poly-lobular.

In contradistinction to this a rarer form has been observed in which ascites is usually absent or slight, jaundice pronounced and continuous; the liver usually enlarged, often greatly so, and the connective tissue arranged around individual lobules, which are thus isolated. In the connective tissue large numbers of bile-capillaries and ducts forming plexuses are seen. Moreover, the connective tissue tends to penetrate the lobules between the rows of liver cells.

These conditions, together with some other clinical features, have led to the establishment of a form of cirrhosis which has received the names of hypertrophic, monolobular, or "biliary" cirrhosis, a form especially characterised by the absence of ascites, presence of very marked jaundice, and enlargement of the liver.

The view has been accepted by some that this form of cirrhosis is due to obstruction of the bile-ducts. Partial changes of a similar nature have been observed where a bile-duct has been obstructed by new growth or by calculus or stricture. Moreover, Charcot succeeded in producing a similar change in rabbits by ligature of the common bile-duct.

These, and many other observations which I must not here detail, have led to the view that this form of cirrhosis is especially related to irritation of the bile-ducts (hence the name "*biliary*" cirrhosis), and most frequently caused by their obstruction, catarrhal or other.

The observations here made are then of value as showing that this form of cirrhosis may exist to a most extreme degree *without any obstruction of the bile-ducts*, for they were injected with the greatest

case, and the orifice of the duct in the duodenum was free. Moreover, there was a total absence of *jaundice*, and there was in both cases, as the most obvious result, great *ascites*.

I therefore offer them as a contribution to the general pathology of cirrhosis of the liver.

For other facts with regard to this form of cirrhosis, I may refer the reader to the following amongst many papers:—

Cornil,—Arch. de Physiologie, 1874.

Charcot and Gombault, *idem*, 1876, p. 272.

Hanot,—These de Paris, 1875.

Hanot and Schachmann, Arch. de Physiol. 1887, tome ix. p. 1.

ILLUSTRATIONS.

From Drawings by Dr. J. Tatham Thompson.

Fig. 1. Margin of lobule of liver from Case I. showing the newly-formed connective tissue (*a*), containing numerous branching bile-ducts (*b*). The liver cells at the margin of the lobule are seen to be separated, the connective tissue penetrating between them. At *b b* the termination of one of the bile-ducts in a row of liver cells is seen; *c* liver cells.

Fig. 2. Very low power drawing of part of the liver in Case II. The bile-ducts were injected with Berlin blue, but are not all filled. The remains of the lobules and islets of liver cells which have become detached are surrounded by broad zones of connective tissue, containing dense plexuses of newly-formed bile-ducts (*b*). At *h* the central vein of a lobule, somewhat dilated, is visible.

Fig. 3. A portion of the same section seen under a higher power. The anastomosing plexuses of bile-capillaries (*b*) are better seen.

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